

PLACES: Sharon, Windsor, Vt.  
To indicate that a child is an ancestor of the person submitting the sheet, place an "x" behind the number pertaining to that child.

ENTER ALL DATA IN THIS ORDER:  
DATES: 14 Apr 1794

FAMILY  
GROUP  
RECORD

HUSBAND William FERGUSON MD

Born \_\_\_\_\_ Place \_\_\_\_\_

Chr. \_\_\_\_\_ Place \_\_\_\_\_

Marr. \_\_\_\_\_ Place \_\_\_\_\_

Died \_\_\_\_\_ Place \_\_\_\_\_

Bur. \_\_\_\_\_ Place \_\_\_\_\_

HUSBAND'S FATHER \_\_\_\_\_ HUSBAND'S MOTHER \_\_\_\_\_

HUSBAND'S OTHER WIVES \_\_\_\_\_

Husband William FERGUSON

Wife Katherine STIRK

Ward Examiner \_\_\_\_\_

Stake or Mission \_\_\_\_\_

WIFE Katherine STIRK MD

Born \_\_\_\_\_ Place \_\_\_\_\_

Chr. \_\_\_\_\_ Place \_\_\_\_\_

Died \_\_\_\_\_ Place \_\_\_\_\_

Bur. \_\_\_\_\_ Place \_\_\_\_\_

WIFE'S FATHER \_\_\_\_\_ WIFE'S MOTHER \_\_\_\_\_

WIFE'S OTHER HUSBANDS \_\_\_\_\_

SEX M F	CHILDREN		WHEN BORN			WHERE BORN			DATE OF FIRST MARRIAGE TO WHOM
	List each child (whether living or dead) in order of birth		DAY	MONTH	YEAR	TOWN	COUNTY	STATE OR COUNTRY	
Given Names	SURNAME								
1									
2									
3									
4									
5									
6									
7									
8									
9									
10									
11									

SOURCES OF INFORMATION

OTHER MARRIAGES

NECESSARY EXPLANATIONS



**Katherine Stirk  
Ferguson M.D.**  
*announces the  
opening of a*  
**Practice Limited  
to Pediatrics**  
**(Birth to 18 Years)**  
  
95 South 500 East  
Heber City  
  
By Appointment  
654-3146

## Brief Report

# Acquired Learning Problems Secondary to Migraine

KATHERINE S. FERGUSON, M.D.

*Department of Pediatrics, The University of Utah Medical Center*

SALLY S. ROBINSON, M.D.

*Department of Pediatrics, University of Texas Medical Branch*

**ABSTRACT.** A case report of a child with severe complicated migraine is presented. Serial psychometric data revealed deterioration of specific cognitive and motor skills and marked behavior change. Early onset of migraine is proposed as a potential cause of learning and behavior problems in some children.

Complicated migraine can result in a variety of permanent neurological deficits.<sup>1,2</sup> Cerebral blood flow studies in migraine patients demonstrate focal reductions in flow sufficient to cause ischemic injury.<sup>3</sup> Areas thought to be ischemic infarcts and focal edema correlating with the neurologic deficit have been seen on CT scans of migraine patients.<sup>4</sup> The association of migraine and stroke is well known. Loss of cognitive skills and behavior changes occur in children secondary to head trauma, infection, cerebrovascular accidents, and other intracranial pathology. Complicated migraine, however, has not been implicated as a cause of learning disorders and behavioral abnormalities in a previously normal child. We present such a case documented by serial psychometric evaluation.

### CASE REPORT

C.W. was a healthy white girl whose medical history was significant only for a strong family history of migraine. She was a superior (all A) student who at ages 10 and 11 years, as a control in psychological study, had functioned in the superior range on the WISC-R. At 13 years she began having episodic difficulty with speech and "weak" legs followed by nausea and bilateral pounding headaches which were relieved by sleep. A diagnosis of migraine was made.

Over the next 10 months she exhibited a variety of transient neurological phenomena, including dysphasia, dysarthria, visual and auditory hallucinations, variations in personality, asymmetric and abnormal reflexes, and loss of voluntary motor

control. Headaches, nausea, and photophobia accompanied most of these attacks.

On three occasions she abruptly became unresponsive except to pain, was restless, combative, and vomiting. These episodes of coma lasted 3 to 5 days with recovery over a week. Emotional lability, immaturity, and poor attention span characterized the convalescent phase, with return to normal behavior between attacks. Primidone, phenobarbital, phenytoin, propranolol HCL and, acutely, hyperbaric oxygen and IV anticonvulsants failed to control the symptoms.

Physical examination remained normal except for the transient neurologic deficits. Intracranial evaluation included several computerized tomography scans with contrast, a pneumoencephalogram, and an arteriogram, all of which were normal. Spinal fluid protein was repeatedly elevated during attacks and once a mild pleocytosis (12 WBC) was noted. With decreased level of consciousness, the electroencephalograms were diffusely and profoundly slow, returning to normal between episodes. Screening for infectious processes, metabolic disorders and intoxication was negative.

Psychometric evaluation was performed 1 month after onset of migraine symptoms, a week after the first episode of coma. She was taking primidone. The examination included Bender Gestalt, Continuous Recognition Memory, Selective Reminding Test, Wide Range Achievement Test, and detailed neurologic evaluation. Behavior was age-appropriate, and intelligence appeared above average. Attention and inhibition of extraneous stimuli were normal as measured by a paced serial addition task. Achievement in reading and spelling were slightly above grade level, but arithmetic was 1½ grade levels below expectations. Learning and retention of new material as well as long range memory were intact. Visual-motor coordination and speed on trailmaking and pegboard tests were normal. Fine motor function and sensation were intact. Writing from dictation and conversational speech were unimpaired.

Nine months later, following the third severe attack, her

Address for reprints: K. S. Ferguson, M.D., Department of Pediatrics, The University of Utah Medical Center, 50 North Medical Drive, Salt Lake City, Utah 84132.